L8 ANSWER 4 OF 24 MEDLINE
AN 2001016572 MEDLINE
DN 20335973 PubMed ID: 10879626
TI Delayed cardioprotection in a **human cardiomyocyte** -derived **cell line**: the role of adenosine, p38MAP kinase and mitochondrial KATP.
AU Carroll R; Yellon D M
CS The Hatter Institute, Department of Academic and Clinical Cardiology, University College Hospitals and Medical School, London, UK.
SO BASIC RESEARCH IN CARDIOLOGY, (2000 Jun) 95 (3) 243-9.
Journal code: 0360342. ISSN: 0300-8428.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001107
AB Evidence of delayed preconditioning (PC) in man is limited. Adenosine is proposed as a trigger via action on the A1 receptor in many species and the mitochondrial KATP channel is a likely end effector. We examined the ability of a brief, simulated ischemic episode on day one to provide delayed cardioprotection against lethal, simulated ischemia on day two in a **human cardiac cell line** with reference to the role of adenosine, the p38MAP kinase signalling pathway and mitochondrial KATP channel. RESULTS: PC and adenosine administered on day 1 protected against cell death on day 2 as measured by LDH release and propidium iodide (PI) exclusion: (%LDH release: PC: 12.1 +/- 1.1%, ADO: 11.9 +/- 2.0% vs control: 36.4 +/- 1.1%; %PI positive: PC: 14.6 +/- 1.4%, ADO: 17.9 +/- 2.0% vs control: 34.4 +/- 2.0% respectively). This protection is abolished by treatment with SB203580 prior to the protective stimulus on day 1: [PC + SB (%LDH release 28.6 +/- 2.8%; %PI positive 34.7 +/- 2.2%) and ADO + SB (%LDH release 25.3 +/- 2.9%; %PI positive 33.7 +/- 7.3)]. Similarly 5-hydroxydecanoate abolished protection, when given immediately prior to lethal simulated ischemia on day 2: [PC + 5-HD; (%LDH release 31.9 +/- 4.8%; %PI positive 29.5 +/- 2.0%) and ADO + 5-HD (%LDH release 36.9 +/- 4.0%; %PI positive 34.8 +/- 2%)]. CONCLUSION: In this model delayed PC can be mimicked by adenosine and involves the p38MAP kinase pathway and the mitochondrial KATP channel.

L8 ANSWER 6 OF 23 MEDLINE
AN 2002350140 MEDLINE
DN 22088243 PubMed ID: 12094073
TI Molecular characterization of regenerated **cardiomyocytes** derived from adult mesenchymal stem cells.
AU Fukuda Keiichi
CS Institute for Advanced Cardiac Therapeutics, Keio University School of Medicine, Tokyo 160-8582, Japan.. kfukuda@sc.itc.keio.ac.jp
SO Congenit Anom Kyoto, (2002 Mar) 42 (1) 1-9.
Journal code: 9306292. ISSN: 0914-3505.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200208
ED Entered STN: 20020703
Last Updated on STN: 20020813
Entered Medline: 20020812
AB We recently isolated a cardiomyogenic (CMG) cell line from murine bone marrow stroma, and in this paper characterize regenerated **cardiomyocytes** derived from adult mesenchymal stem cells at the molecular level. **Stromal** cells were immortalized, exposed to 5-azacytidine, and repeatedly screened for spontaneously beating cells. CMG cells began to beat spontaneously after 2 weeks, and beat synchronously after 3 weeks. They exhibited sinus-node-like or ventricular-cell-like action potentials. Analysis of the isoforms of contractile protein genes, such as of myosin and alpha-actin, indicated that their phenotype was similar to that of fetal ventricular **cardiomyocytes**. The cells expressed Nkx2.5, GATA4, TEF-1, and MEF2-C mRNA before 5-azacytidine exposure, and MEF2-A and MEF2-D after exposure. CMG cells expressed alpha1A, alpha1B, and alpha1D-adrenergic receptor mRNA prior to differentiation, and beta1, beta2-adrenergic and M1, M2-muscarinic receptors after acquiring the **cardiomyocyte** phenotype. Phenylephrine induced phosphorylation of ERK1/2, and the phosphorylation was inhibited by prazosin. Isoproterenol increased the cAMP level 38-fold and beating rate, cell motion, %shortening, and contractile velocity by 48%, 38%, 27%, and 51%, respectively, and the increases were blocked by CGP20712A (beta1-selective blocker). Carbachol increased IP3 32-fold, and the increase was inhibited by AFDX116 (M2-selective blocker). These findings demonstrated that the regenerated **cardiomyocytes** were capable of responding to adrenergic and muscarinic stimulation. This new cell line provides a model for the study of **cardiomyocyte** transplantation.

L8 ANSWER 10 OF 24 MEDLINE
AN 97304504 MEDLINE
DN 97304504 PubMed ID: 9160867
TI Dedifferentiated **human** ventricular **cardiac myocytes** express inducible nitric oxide synthase mRNA but not protein in response to IL-1, TNF, IFNgamma, and LPS.
AU Luss H; Li R K; Shapiro R A; Tzeng E; McGowan F X; Yoneyama T; Hatakeyama K; Geller D A; Mickle D A; Simmons R L; Billiar T R
CS Department of Surgery, University of Pittsburgh School of Medicine, PA 15261, USA.
NC GM-37753 (NIGMS)
GM-44100 (NIGMS)
SO JOURNAL OF MOLECULAR AND CELLULAR CARDIOLOGY, (1997 Apr) 29 (4) 1153-65.
Journal code: 0262322. ISSN: 0022-2828.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF068236
EM 199707
ED Entered STN: 19970724
Last Updated on STN: 20000303
Entered Medline: 19970717
AB There is evidence that nitric oxide (NO) may mediate some of the functional myocardial changes caused by bacterial LPS and inflammatory cytokines. The expression of the inflammatory or inducible NO synthase (iNOS) in **human cardiac myocytes**, however, has not been well characterized. Therefore, we treated cultured, dedifferentiated **human** ventricular **cardiac myocytes** with the combination of TNF-alpha (500 U/ml), IL-1beta (30U/ml), IFNgamma (100 U/ml), and LPS (E.coli 0111:B4, 10 microg/ml). Northern blot analysis revealed a approximately 4.5 kb transcript for inducible NOS (iNOS) in the stimulated **human** heart cells but not in untreated cells. RT-PCR confirmed that iNOS mRNA was only present in stimulated cells. However, treatment of the myocytes for up to 96 h with cytokines and LPS did not result in NO synthesis as measured by nitrite + nitrate accumulation in the culture medium, and no iNOS enzymatic activity could be detected in the cell lysates. Western blot analysis failed to detect iNOS protein. Thus, despite high and persistent levels of iNOS mRNA in cytokine-treated cells, iNOS protein was absent in this experimental model. GTP-cyclohydrolase I was induced both at the mRNA and protein levels and resulted in increased biopterin levels, indicating sufficient amounts of the cofactor tetrahydrobiopterin (BH4) were present, and that the failure to express an inducible protein was specific to iNOS. To determine if the absence of iNOS protein was due to a novel cardiac iNOS gene or modified iNOS transcript in **human** myocytes, we cloned an iNOS cDNA from cytokine-treated myocytes. Sequencing and expression of the clone revealed a functional iNOS cDNA with >99% identity to other **human** iNOS cDNA clones. When **human cardiac** cells were transduced with a retroviral vector carrying only the coding region of the **human** hepatocyte iNOS cDNA, both iNOS mRNA and protein could be detected. In conclusion, these cells derived from cultured **human cardiac myocytes** lacked the capacity to express an endogenous iNOS protein, the basis of which appears to be a cell-specific suppression or failure of iNOS translation.

L1 ANSWER 6 OF 11 MEDLINE
AN 1999175236 MEDLINE
DN 99175236 PubMed ID: 10074487
TI **Cardiomyocytes** can be generated from marrow stromal cells in vitro.
AU Makino S; Fukuda K; Miyoshi S; Konishi F; Kodama H; Pan J; Sano M; Takahashi T; Hori S; Abe H; Hata J; Umezawa A; Ogawa S
CS Cardiopulmonary Division, Department of Internal Medicine, Keio University School of Medicine, Tokyo 160-8582, Japan.
SO JOURNAL OF CLINICAL INVESTIGATION, (1999 Mar) 103 (5) 697-705.
Journal code: 7802877. ISSN: 0021-9738.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199903
ED Entered STN: 19990413
Last Updated on STN: 19990413
Entered Medline: 19990331
AB We have isolated a cardiomyogenic cell line (CMG) from murine bone marrow stromal cells. Stromal cells were **immortalized**, treated with 5-azacytidine, and spontaneously beating cells were repeatedly screened. The cells showed a fibroblast-like morphology, but the morphology changed after 5-azacytidine treatment in approximately 30% of the cells; they connected with adjoining cells after one week, formed myotube-like structures, began spontaneously beating after two weeks, and beat synchronously after three weeks. They expressed atrial natriuretic peptide and brain natriuretic peptide and were stained with anti-myosin, anti-desmin, and anti-actinin antibodies. Electron microscopy revealed a **cardiomyocyte**-like ultrastructure, including typical sarcomeres, a centrally positioned nucleus, and atrial granules. These cells had several types of action potentials, such as sinus node-like and ventricular cell-like action potentials. All cells had a long action potential duration or plateau, a relatively shallow resting membrane potential, and a pacemaker-like late diastolic slow depolarization. Analysis of the isoform of contractile protein genes, such as myosin heavy chain, myosin light chain, and alpha-actin, indicated that their muscle phenotype was similar to that of fetal ventricular **cardiomyocytes**. These cells expressed Nkx2.5/Csx, GATA4, TEF-1, and MEF-2C mRNA before 5-azacytidine treatment and expressed MEF-2A and MEF-2D after treatment. This new cell line provides a powerful model for the study of **cardiomyocyte** differentiation.

L1 ANSWER 5 OF 11 MEDLINE
AN 2000074319 MEDLINE
DN 20074319 PubMed ID: 10608607
TI Ag+ alters cell growth, neurite extension, **cardiomyocyte** beating, and fertilized egg constriction.
AU Conrad A H; Tramp C R; Long C J; Wells D C; Paulsen A Q; Conrad G W
CS Division of Biology, Kansas State University, Manhattan 66506-4901, USA. aconrad@ksu.edu.
SO AVIATION SPACE AND ENVIRONMENTAL MEDICINE, (1999 Nov) 70 (11) 1096-105. Journal code: 7501714. ISSN: 0095-6562.
(Investigators: Spooner B S, KS St U, Manhattan)
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 200001
ED Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000107
AB BACKGROUND: The Russian Space Agency uses electrochemically generated silver ions (Ag+) to purify drinking water for their space station, Mir, and their portion of the International Space Station. U.S. EPA guidelines allow 10.6 micromol x L(-1) Ag+ in human drinking water for up to 10 d. Studies correlate Ag+ exposure with tissue dysfunction in humans, rats, and mice, and with altered ion transport, skeletal muscle contraction, and embryonic cell constriction in other animal cells. Ag+ effects on cell shape change-related functions have not been assessed. METHODS: **Immortalized** embryonic human intestinal epithelial cells, freshly explanted embryonic avian nerve cells and **cardiomyocytes**, and marine fertilized eggs were grown in vitro in medium containing AgNO3. RESULTS: Intestinal cells detach from the substratum and viable cell number decreases by 5-6 d at 5 micromol x L(-1) AgNO3, and faster at higher concentrations. Microtubules appear unaltered in adherent cells. Detached cells are nonviable. Neurite outgrowth and glial cell migration from dorsal root ganglia are inhibited by 3 d at 15 micromol x L(-1) AgNO3 or greater. Contractions stop temporarily in most **cardiomyocytes** by 5 min at 5 micromol x L(-1) AgNO3 or more, but some **cardiomyocytes** beat 3 times faster than normal at 7.5-20 micromol x L(-1) AgNO3. Picomolar Ag+ increases marine egg polar lobe constriction within an hour, even in the absence of microtubules. CONCLUSION: Ag+ alters animal cell growth and shape changes by a MT-independent mechanism. This is the first report of Ag+ effects on vertebrate neurite outgrowth, glial cell migration, or **cardiomyocyte** beat rate.

09/604,876

```
=> s primary mitotic cells
        426463 PRIMARY
          1156 PRIMARIES
        426853 PRIMARY
                 (PRIMARY OR PRIMARIES)
         27029 MITOTIC
             8 MITOTICS
         27033 MITOTIC
                 (MITOTIC OR MITOTICS)
       1380872 CELLS
L4           0 PRIMARY MITOTIC CELLS
                 (PRIMARY(W)MITOTIC(W)CELLS)

=> s primary post-mitotic cells
        426463 PRIMARY
          1156 PRIMARIES
        426853 PRIMARY
                 (PRIMARY OR PRIMARIES)
        203700 POST
          1487 POSTS
        204616 POST
                 (POST OR POSTS)
         27029 MITOTIC
             8 MITOTICS
         27033 MITOTIC
                 (MITOTIC OR MITOTICS)
       1380872 CELLS
L5           0 PRIMARY POST-MITOTIC CELLS
                 (PRIMARY(W)POST(W)MITOTIC(W)CELLS)

=> s primary culture
        426463 PRIMARY
          1156 PRIMARIES
        426853 PRIMARY
                 (PRIMARY OR PRIMARIES)
        341031 CULTURE
        143120 CULTURES
        423626 CULTURE
                 (CULTURE OR CULTURES)
L6       16974 PRIMARY CULTURE
                 (PRIMARY(W)CULTURE)

=> s l1 and l6
L7           26 L1 AND L6

=> d l7 ibib abs total

L7   ANSWER 1 OF 26     MEDLINE
ACCESSION NUMBER:   2001242267     MEDLINE
DOCUMENT NUMBER:    21243036   PubMed ID: 11344338
TITLE:              MK/T-1, an immortalized fibroblast cell
                    line derived using cultures of mouse corneal
                    stroma.
AUTHOR:             Gendron R L; Liu C Y; Paradis H; Adams L C; Kao W W
CORPORATE SOURCE:   Department of Pediatrics, Division of Hematology and
                    Oncology, Children's Hospital Medical Center, University of
                    Cincinnati, Cincinnati, OH 45229-3039, USA..
                    rlgendron@chmcc.org
CONTRACT NUMBER:    EY10556 (NEI)
                    EY11845 (NEI)
                    EY12486 (NEI)
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(immortalized human cardiomyocyte)	264652

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US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Refine Search: (immortalized human cardiomyocyte) Clear

Search History

Today's Date: 2/4/2002

DB Name	Query	Hit Count	Set Name
USPT	(immortalized human cardiomyocyte)	264652	L4
USPT	(immortalized human cardiomyocyte)	264652	L3
USPT	(immortalized cell line and human cardiomyocyte)	454398	L2
USPT	(immortalized cell line near4 human)	362070	L1